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CASE REPORT

Pituitary Apoplexy: Pitfalls in Diagnosis

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Abstract

Headache is a common presenting complaint in the emergency department. A rare cause is pituitary apoplexy − a complication of pituitary adenoma consisting of hemorrhage or infarction of the primary tumor accounting for approximately 1 % of headaches. A 44-year-old female presented with intractable headache, nausea, photophobia and later − signs of meningeal irritation. Initial imaging demonstrated no mass or hemorrhage, labs showed only leukocytosis and elevated CRP. Patient was started on empiric acyclovir and methylprednisolone. CSF analysis was negative for meningitis, thus MRI of the brain was performed which demonstrated a 2.5 cm suprasellar mass. Initial Pituitary hormone evaluation demonstrated low prolactin, normal TSH and low ACTH thought to be due to steroid use. Repeat laboratory evaluation demonstrated hypopituitarism. Patient underwent resection of the adenoma with pathology consistent with pituitary apoplexy. We highlight the need for careful evaluation of patients presenting with headache and signs of meningeal irritation given 16 % prevalence of pituitary adenoma. CT of the head may not always demonstrate acute infarction, with MRI of the brain remaining the most sensitive imaging modality. Given the common use of methylprednisolone for headache, a pitfall in the diagnosis of pituitary apoplexy includes proper assessment of a pituitary panel prior to initiation of steroids.

Keywords: Pituitary apoplexy, Headache, Pituitary adenoma, Subarachnoid hemorrhage

1. Introduction

Headache is a common presentation in the emergency department with multiple possible etiologies ranging from benign to life-threatening. Primary headache accounts for 45 % of cases, with migraine diagnosed in 23 %.[1,2] Approximately, 7 % of presenting headaches are due to secondary causes. One such cause is pituitary apoplexy (PA) − a rare complication of pituitary adenoma accounting for 1 % of presenting headaches, resulting from infarction or subarachnoid hemorrhage of the primary tumor. Headache is the most prominent symptom, present in over 80 % of patients, usually described as sudden and severe and is thought to be due to blood extravasation and dural traction with resultant meningeal irritation. Over 50 % of patients also experience visual disturbances, the most common one being bitemporal hemianopsia, as well as photophobia, nausea, meningismus and fever. Because of the nonspecific symptoms, diagnosis and treatment can be delayed resulting in negative outcomes.

2. Case presentation

A 44-year-old female presented to our institution with a one-day history of intractable headache associated with nausea, vomiting, photophobia and phonophobia. The headache was located in the occipital region, radiating to the front. The patient reported a past medical history of left hemithyroidectomy for thyroid nodules, obesity, GERD, prediabetes, and hirsutism. She had no prior history of migraines. On initial evaluation, patient reported tenderness to palpation of the trapezius muscles, however Kernig and Brudzinski signs were negative. On the second day of admission, she started developing signs of meningeal irritation, prompting further evaluation by neurology.

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3. Diagnostic assessment

Initial labs were unremarkable except for mild leukocytosis and elevated CRP. CSF evaluation was negative for infection, xanthochromia or RBC elevation. TSH drawn on admission was low-normal (0.69uIU/mL). Initial pituitary hormone evaluation on hospital day 4 demonstrated a low TSH (0.259 uIU/mL) and a free T4 of 0.78 ng/dL. Serum AM Cortisol was normal at 10.8ug/dL and ACTH was low (<1.5 pg/mL), however it was thought to be suppressed due to methylprednisolone use.[3] Prolactin levels were also low (0.3 ng/mL) raising suspicion for pituitary apoplexy. Non-contrast CT scan of the head on admission revealed no mass or signs of intracranial hemorrhage. CTA was negative for thrombotic or vasculitic etiologies. Due to persistent headache, an MRI was performed. It revealed a 2.5 cm sellar mass with suprasellar extension abutting the optic chiasm consistent with pituitary adenoma (Fig. 1).

4. Treatment

Patient was initially started on empiric acyclovir, which was discontinued after negative CSF analysis. Pulse dose methylprednisolone was started concomitantly for potential differentials of viral meningitis or status migrainosus. After discovery of the pituitary mass, neurosurgery was consulted with a recommendation for outpatient follow-up and pituitary hormone workup. Due to improvement in headache, patient was discharged on hospital day 6.

5. Outcome and follow-up

One week after the last steroid dose, repeat labs demonstrated low TSH (0.058 uIU/mL), low Free T4 (0.49 ng/dL), low prolactin (0.6 ng/mL), low ACTH (3.7 pg/mL) and low morning cortisol (0.8 ug/dL) which was suggestive of secondary adrenal insufficiency and secondary hypothyroidism. The patient also developed fatigue, polyuria and polydipsia,

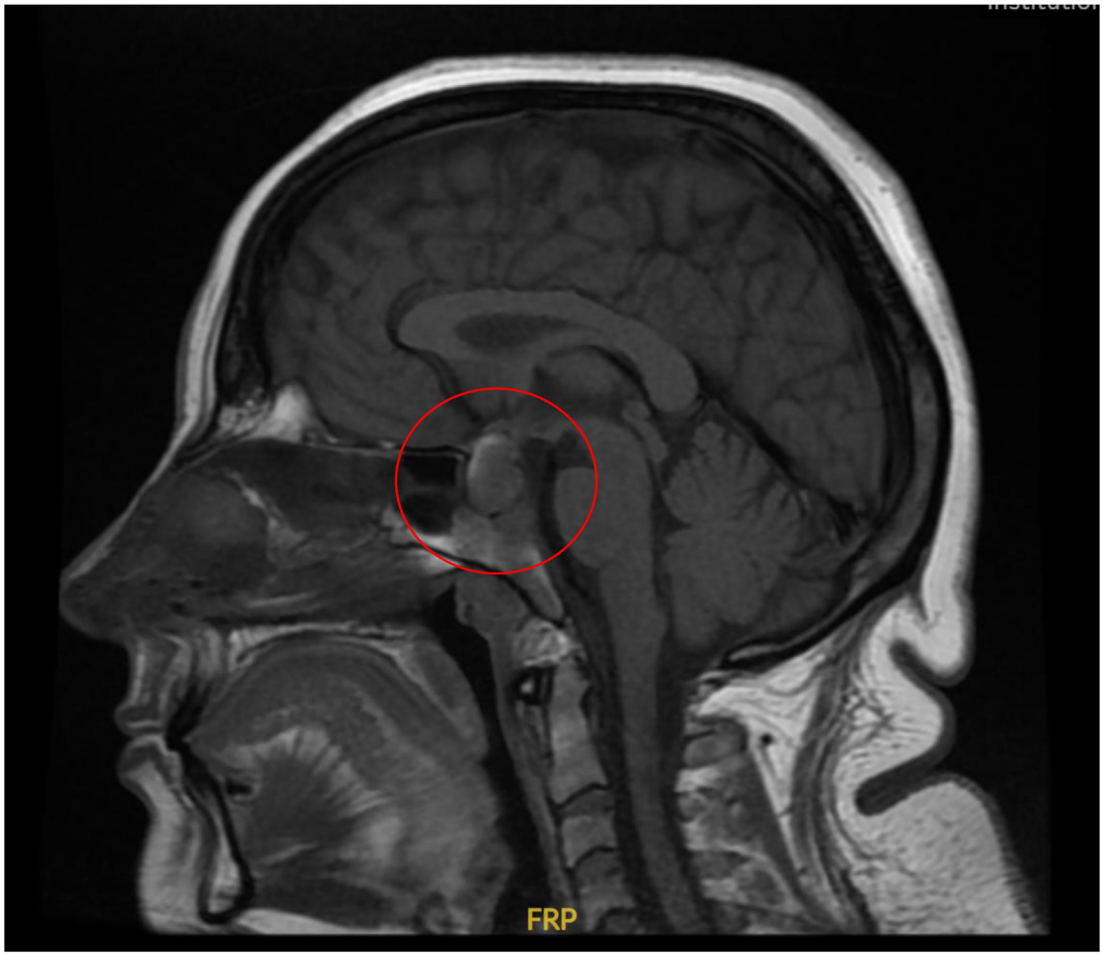

Fig. 1. 2.5 sellar mass abutting the optic chiasm and the proximal pre-chiasmatic segment of the bilateral optic nerves, consistent with a macroadenoma.

symptoms highly suggestive of panhypopituitarism. Neurosurgery performed transsphenoidal resection 24 days after initial presentation and surgical pathology showed necrotic tissue confirming the diagnosis of pituitary apoplexy. Patient is currently maintained on hormone replacement therapy with hydrocortisone, DDAVP and levothyroxine. Patient is followed by endocrinology, with routine laboratory testing and adjustments in her regimen as appropriate. Repeat outpatient MRI has demonstrated no evidence of residual tissue or recurrent disease.

6. Discussion

Pituitary adenomas have a prevalence of 16.7 % with 2–12 % of all patients developing pituitary apoplexy.[4] This must be differentiated from other etiologies of headache such as meningitis, brain masses/space-occupying lesions, subarachnoid hemorrhage, and primary headache.[1] Given the prevalence of pituitary adenoma in the general population, the possibility of pituitary apoplexy should be contemplated as it is a medical emergency. One if its major complications is blindness which has an incidence of 35–42 %.[5] As a result, prompt diagnosis is key and patient headache are often initially misdiagnosed and treated with a variety of medications, including steroids. These can mask developing secondary adrenal insufficiency and secondary hypothyroidism, given the inhibitory effect of steroids on TRH.[6] Initial workup will demonstrate low prolactin and ACTH levels which reveal underlying pituitary apoplexy and help differentiate it from other etiologies of headache. Therefore, if suspecting pituitary apoplexy, obtaining a pituitary panel with initial blood work should be considered. Computer tomography is the first imaging modality that patients undergo when presenting at the emergency department, however it has low sensitivity ranging from 21 to 46 % for diagnosing pituitary apoplexy, thought to be due to the evolution of the hemorrhage and degradation of blood products.[7] MRI has a sensitivity of 88–99 % and is considered the best imaging modality for diagnosing pituitary apoplexy.[7] Even though Pituitary adenomas account for approximately 1 % of all acute headache presentations, given the high prevalence of pituitary adenoma in the general population, clinicians should consider this diagnosis as part of their differential for patients presenting with acute headache, particularly when associated with visual deficits and signs of meningeal irritation.

Learning points

- On assessing headache, with visual symptoms and meningeal signs, pituitary apoplexy should be high in the differential diagnoses as it is a medical emergency and is more prevalent than expected.
- Laboratory evaluations including a pituitary panel should be drawn before administration of medications such as steroids to avoid alterations in values.
- Prompt MRI should be considered given its higher sensitivity for diagnosing pituitary apoplexy.

Disclaimer

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